

Industrial bronchitis

W. K. C. MORGAN¹

From the West Virginia University Medical Center, Morgantown, WV 26506, USA

ABSTRACT For many years there has been much argument whether workers in the dusty trades are prone to chronic bronchitis. In 1966 the Medical Research Council issued a report of a Select Committee which concluded that occupationally induced bronchitis did not play a significant part in the aetiology of airways obstruction in dust-exposed men. Since then epidemiological studies have demonstrated that the prolonged inhalation of dust leads to an increase in prevalence of cough and sputum. Furthermore, new physiological techniques have demonstrated a slight decrement in ventilatory capacity as a result of industrial bronchitis, and which is related to lifetime dust exposure. Unlike bronchitis induced by cigarette smoke, the predominant effect of industrial bronchitis is on large rather than small airways and the condition is not accompanied by emphysema.

An increased prevalence and high death rate from bronchitis were recognised in certain occupations in the nineteenth century by Thackrah (1832) and Greenhow (1861). However, the term bronchitis as used by these authors was not specific and the symptoms that were attributed to the condition were equally likely to have been caused by other chest diseases. It was not until halfway through the present century that it became possible to elucidate the respective roles of pneumoconiosis, tuberculosis, emphysema, and bronchitis in the production of respiratory symptoms in the dusty trades. Even now there is much debate about the actual existence of industrial bronchitis and about its possible effects on ventilatory capacity and life expectancy.

The term industrial bronchitis is used here to denote a condition characterised by cough and sputum for at least three months a year, with or without airways obstruction, and which is related to the long-continued inhalation of dust. It is clearly important to define the role of industrial bronchitis in the pathogenesis of occupationally related lung disease, for several reasons. First, it has been suggested that much of the increased standardised mortality ratio (SMR) that has been reported in the dusty occupations is a result of chronic bronchitis (Lowe, 1968). Second, the observation in coal miners that there is a poor relationship between pneumoconiosis and the presence of respiratory symptoms of bronchitis suggests that the aetiology of the two conditions may

be different (Gilson, 1970). Third, because most preventive measures, and in particular dust standards, are directed at the control of pneumoconiosis rather than of industrial bronchitis, if the latter indeed had a significant effect on pulmonary function, present dust control methods might well prove to be ineffective or deficient.

In 1966 the Medical Research Council (MRC) published a committee report entitled 'Chronic Bronchitis and Occupation'. The stimulus to the appointment of this committee was the concern expressed by the then Minister of Pensions and National Insurance in that, while coalminers with pneumoconiosis and bronchitis were eligible for insurance benefits, those with a clear chest film were not. The committee was charged with examination of the relationship between bronchitis and occupation. Nowhere in the statement does the committee define chronic bronchitis, and one is left with the impression that in some instances the term is synonymous with cough and sputum, while elsewhere cough, sputum and ventilatory obstruction are implied. They concluded that 'on present evidence intensity of dust exposure does not appear to be a very significant factor in determining the presence of bronchitis in groups of workers'. Their reasons for this conclusion were as follows: first, the symptoms of chronic bronchitis, namely, cough and sputum, are the same whatever the cause. It was therefore not possible to apportion the contributions of cigarette smoking, air pollution, occupational exposure, or other environmental conditions to the development of bronchitis. The committee then went further to say, 'even if there were incontrovertible evidence that bronchitis was higher among workers

¹Now Professor of Medicine, University of Western Ontario, and Medical Director, Sir Adam Beck Chest Diseases Unit, University Hospital, London, Ontario, Canada.

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in certain occupations than in others, it would not be possible on the basis of any known form of clinical examination to measure the extent to which the disease in any individual was due to his occupation'. Second, epidemiological data relating bronchitis to morbidity and mortality, although suggesting an occupational component, were subject to distorting biases. It was pointed out that the Registrar General's statistical surveys were, and continue to be, based on the number of men with the condition among those currently doing a specific job compared with the total number of men thus employed at the same time of the census. It was suggested that, as the statistics usually reflect the man's present job, while death certificates usually reflect the man's original job or the job at which he has worked for most of his life, these factors tend to inflate the number of miners reported as face workers, at least as far as death certification is concerned. Industrial migration was also felt to pose a serious problem in the interpretation of mortality and morbidity statistics. Other anomalies also existed: for example, in the Registrar General's statistics, bronchitis was, and continues to be, a far more frequently certified cause of death in the lower socioeconomic groups; this phenomenon was particularly evident in coalminers who had worked at the coal face. However, a comparable increase in the SMR was also present for the wives of miners, and it is difficult to incriminate occupation as a causative factor in the latter. Similarly, field surveys had shown a higher prevalence of bronchitis in miners' wives than in wives of nonminers. Nonetheless, the committee conceded that miners in South Wales, but not elsewhere, appeared to have a higher incapacity rate for bronchitis than could be attributed to air pollution alone.

The conclusions of the MRC committee were hotly disputed, but the arguments used to refute the MRC's statement seemed based for the most part on clinical impressions and sympathy rather than objective data (McLaughlin, 1966; Pemberton, 1966). Nevertheless, it was pointed out that, in the Rhondda Fach, while certified deaths from bronchitis for men were greatly increased above those for the general population, this was not true for women (Gough, 1966).

When compared with a control population, a significant increase in the prevalence of cough and sputum has been observed in many men working in dusty occupations, including coalminers (Higgins *et al.*, 1959; Worth *et al.*, 1959; Higgins and Cochrane, 1961; Higgins, 1972), steel workers (Lowe *et al.*, 1970), textile manufacturers (Merchant *et al.*, 1972), gold miners (Sluis-Cremer *et al.*, 1967), and cement workers (Kalacić, 1973). In some in-

stances a slight reduction in ventilatory capacity has been observed to accompany the increased prevalence of cough and sputum. However, these observations, especially with regard to coalminers, have not been consistent and anomalous findings have been fairly frequent (Enterline, 1967; Higgins, 1972; Minnette, 1976). Although some of these facts were known at the time of the MRC report, the committee made no reference to them;

The presence of the reduced ventilatory capacity that has been observed in coalminers without progressive massive fibrosis can be explained by two hypotheses:

1. that there is a type of airflow obstruction, peculiar to dusty trades, which affects miners with and without pneumoconiosis. The cause of this obstruction could be either bronchitis or emphysema or both;
2. that it is due to differential migration; thus, were men with better lung function to move out of the industry within a few months or years of starting work, those who remain would have substantially reduced pulmonary function and would be a non-representative sample of the local population.

The second hypothesis must be considered because Cochrane *et al.* (1961) have adduced evidence to suggest that, at times when unemployment is high, it is the fittest who tend to migrate and seek new jobs elsewhere. However, a study by McLintock (1971) of new entrants into mining showed that those who leave first tend to be less fit and more often have chest symptoms. The very fit tend to work on and this, perhaps, explains why miners with radiological category 3 simple pneumoconiosis often have a higher ventilatory capacity than do those with lower categories.

The relationship of obstructive emphysema to dust exposure

With regard to the first hypothesis, it has recently been suggested that coalminers have a higher incidence of emphysema than do nonminers. The bases for this contention have been published in a series of papers from South Wales (Ryder *et al.*, 1970; Lyons *et al.*, 1972; Lyons and Campbell, 1976). These studies relate the post-mortem findings in over 250 Welsh miners, with particular emphasis on emphysema, to ante-mortem physiological and radiological data. The presence and extent of emphysema were quantified by 'point counting'. The authors found a relationship between the presence of simple and complicated pneumoconiosis and ante-mortem evidence of ventilatory impairment as diagnosed by a reduced forced expiratory volume

in one second ($FEV_{1.0}$). The presence of 'p' or punctate type opacities seen in radiographs of simple pneumoconiosis of coal workers was felt to be more commonly associated with emphysema than the larger rounded opacities (q and r). The first paper contained the statement that 'There is no reason why these deaths should not provide a true sample of the experience of men with this disease.' In short, it was suggested that the sample was representative of miners and ex-miners not only in Wales, but elsewhere. This contention was disputed by Gilson and Oldham (1970) who pointed out that all the subjects included in the series had been awarded disability benefit, and therefore were likely to differ appreciably from the majority of miners who had not. Moreover, in the first paper, Ryder *et al.* (1970) attempted to relate the presence of post-mortem emphysema to the degree of pneumoconiosis diagnosed from the last radiographic examination before death. No distinction was made between subjects with simple and complicated disease, and because the latter is known to be associated with airways obstruction, this again could lead to spurious results. In the second paper the authors did not separate subjects with simple pneumoconiosis from those with complicated (PMF) and again the same issue was raised (Oldham and Berry, 1972). There are other anomalies in their results, in particular the observation that miners with categories 2 and 3 simple pneumoconiosis had an $FEV_{1.0}$ which was 0.8 litres below the predicted value, while those with category 0 had an $FEV_{1.0}$ that was 1.25 litres below the predicted value.

Seaton *et al.* (1972) compared the ventilatory capacity, lung volumes, and diffusing capacity with the type of radiographic opacity (p,q,r) in a large group of working miners in the United States of America. Apart from a slight reduction in diffusing capacity in miners with the p type, no other differences were noted. If more emphysema were present in miners with the p type of opacity, then the residual volume and total lung capacity should have been increased in comparison with miners having the q and r type of opacity, but this was not so. At about the same time, Waters *et al.* (1974) showed that the p type of opacity was not associated with decreased longevity, a fact which is surprising if indeed emphysema and a lower $FEV_{1.0}$ were present more often in miners with the p type of simple pneumoconiosis. Thus, the weight of the evidence seems to be against emphysema being responsible for the lower ventilatory capacity that has been noted in coalminers.

Bronchitis and dust exposure

The Medical Research Council (1966) statement,

which implied that bronchitis was a single response to a variety of insults, most of which could not be identified, at least in the individual, was probably justified at the time it was published. However, the committee's addendum 'nor is there any reason to believe that it will at some time be possible to determine in any particular individual with chronic bronchitis how much of his or her illness is attributable to any particular environmental factor' seems singularly pessimistic, and in the light of recent investigations was obviously erroneous. Ten to twenty years ago the term 'bronchitis' implied a condition characterised by cough and sputum and usually associated with a reduction in ventilatory capacity. While it was realised that not all subjects with cough and sputum showed a decrement in pulmonary function, it was assumed that in most there was a cause-and-effect relationship between the symptoms and the pulmonary function abnormalities and that, sooner or later, all subjects with cough and sputum would develop ventilatory impairment. This concept has been shown to be incorrect, and the studies of Fletcher and Peto (1977) and Bates (1973) make it clear that bronchitis and airways obstruction are not necessarily related. It is apparent from these studies that bronchitis exists in a simple form, that is, the hypersecretion of mucus in the absence of ventilatory impairment, and as obstructive bronchitis in which cough and sputum are accompanied by a reduction in ventilatory capacity. Moreover, it is now apparent that, although bronchitis and airflow obstruction are both related to cigarette smoking, the presence of bronchitis plays only a minor part in the production of the increased resistance to airflow. Thus, chronic bronchitis is a condition that affects the large airways and is characterised by an excessive amount of mucus. In contrast, it is the peripheral airways, those distal to the 12th generation, that are the anatomical site of the pathological changes that occur in irreversible airways obstruction. This new epidemiological information has a direct bearing on the problems of industrial bronchitis.

While numerous studies have shown that the dusty trades are associated with cough and sputum, only over the past few years has it been shown that there is a definite relationship between the prevalence of these symptoms and the level of dust exposure (Rae *et al.*, 1971). Kibelstis *et al.* (1973) showed that the prevalence of bronchitis in non-smoking coalminers in the United States was related to the degree of dust exposure in that the disease was most common in face workers and least common in surface workers. For the most part, these effects were obvious only in the non-smokers, because cigarette smoking overwhelmed and obscured the

effects of other environmental agents. In other studies, cigarette smoking, dust and other environmental factors appear to have had a synergistic effect (Sluis-Cremer *et al.*, 1967; Minnette, 1976).

It has been much more difficult to show a relationship between reduced ventilatory capacity measured by FEV_{1.0}, and dust exposure. Nonetheless, in the study of Kibelstis *et al.* (1973), a difference in FEV_{1.0} between non-smoking face workers and non-smoking surface workers was apparent. The difference, although statistically significant because of the large number of subjects included, was of little clinical importance (FEV_{1.0} of surface workers 101% of predicted; FEV_{1.0} of face workers 98% of predicted). Long-term studies by the National Coal Board have shown that there is a progressive decrement in FEV_{1.0} with cumulative dust exposure, and, more important, that the presence of simple pneumoconiosis does not have an effect over and above that attributable to dust exposure (Rogan *et al.*, 1973). It is a testimony to the accuracy and worth of the long-term environmental measurements that the National Coal Board has been making, that it has been able to demonstrate this effect. Nonetheless, it is clear that cigarette smoking has about a five to ten times greater effect on ventilatory capacity, as measured by the FEV_{1.0}, than does coal dust.

The FEV_{1.0} and the commonly used tests of ventilatory capacity reflect mainly the behaviour of the upper airways during dynamic compression. Thus, the FEV_{1.0} depends for the most part on the resistance to flow in the trachea, lobar, segmental and subsegmental bronchi. Obstruction in small airways (12th generation and beyond) is difficult to detect with the standard tests of ventilatory capacity, namely the FEV_{1.0} and forced vital capacity (FVC). In addition, about 85% of the total airways resistance (R_A) is located in the larger airways while only about 15% is in the peripheral airways. Hence, it is possible for the resistance in the peripheral airways to double or quadruple, and for the FEV_{1.0} and R_A to remain within the normal range. Recently, several techniques have been introduced which allow physiologists to assess independently the resistance to airflow in the central and peripheral airways (Morgan *et al.*, 1974). These include the determination of dynamic compliance at increasing respiratory rates, measurement of closing volume, determination of alveolar-arterial oxygen gradients and the use of flow-volume curves. While not all of these techniques can be applied in the field, the flow-volume curve has been used successfully in epidemiological studies. A typical expiratory curve is shown in Figure 1. There is strong evidence that peak flow and flow at 25% of vital capacity (FEF₂₅) reflect the state of the large airways. Flow at 50%

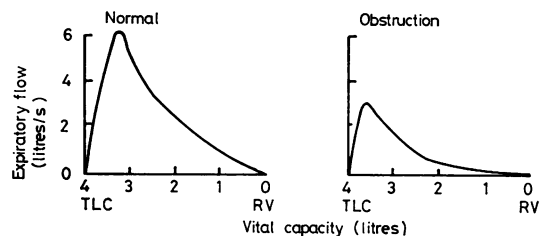


Fig. 1 A normal expiratory flow-volume curve and that of an obstructed subject for comparison

of vital capacity (FEF₅₀) depends on both small and large airways function, while flows at 75% of vital capacity (FEF₇₅) and beyond are governed by the mechanical properties of the lung surrounding the small airways, and by the resistance to flow in the latter (Hyatt and Black, 1973).

Hankinson *et al.* (1977a) have used the flow-volume loop in their epidemiological studies of coalminers in the United States of America. They related flow rates to various factors including age, height, radiological category of pneumoconiosis and years spent working underground. Smoking had been shown previously to affect flow at all lung volumes; however when flow at various lung volumes in the non-smoking bronchitic miners was considered, only peak flow and the FEF₂₅ and FEF₅₀ were affected (Table). Flows at low lung volumes

Table Factors influencing expiratory flow rates in coal miners

Variable	Multiple regression analysis regression coefficients for flow rates from 1696 miners (non-smokers)				
	Peak flow	FEF ₂₅	FEF ₅₀	FEF ₇₅	FEF ₉₀
Age (years)	-0.0278	-0.0197	-0.0366	-0.0358	-0.0139
Height (cm)	0.0339	0.0193	0.0144	0.0125	0.0085
Weight (kg)	0.0132	0.0132	0.0062	-0.0053	-0.0075
Underground experience (years)	-0.0349	-0.0319	-0.0155	-0.0001*	-0.0012*
Constant (litres/second)	3.2875	4.0725	3.3087	1.4550	0.2052

*Not significant

were unaffected and, indeed, there was an anomaly in that the FEF₉₀ of the non-smoking bronchitics was higher than that of the non-bronchitic non-smokers. This was explained by an increase in the residual volume (RV) of the bronchitic non-smokers.

Flow rates should be expressed either as a percentage of total lung capacity or, ideally, at absolute lung volume, because changes in lung volume such as an increase in residual volume will decrease the vital capacity and lead to spuriously high values if the flow rates are expressed as a percentage of FVC.

Subsequent studies by Hankinson *et al.* (1977b) have characterised in greater detail the differences between the physiological impairments that are associated with industrial and cigarette-smoke-induced bronchitis. The methodology used in the Interagency Study of working US coalminers (O'Shea *et al.*, 1970) relied upon the flow-volume curve as a means of assessing ventilatory capacity. In addition to the spirometric measurements, postero-anterior and left lateral chest radiographs were taken, from which total lung capacity was calculated. It was thus possible to express flow rates, not only as a percentage of vital capacity (VC), but also as a percentage of total lung capacity (TLC), and at absolute lung volumes. From the sample of over 9000 coalminers, four age- and height-matched groups were selected according to their smoking habits and according to whether they had bronchitis or not, the latter being diagnosed by the presence of daily cough and sputum for at least three months of the year for two consecutive years (Hankinson *et al.*, 1977). Ex-smokers and subjects with progressive massive fibrosis were excluded. Each group comprised 428 subjects and consisted of:

1. smokers with bronchitis
2. smokers without bronchitis
3. non-smokers with bronchitis
4. non-smokers without bronchitis

It is again apparent (Figure 2) that, while cigarette smoking affects flows at all lung volumes, industrial bronchitis predominantly affects peak flow and flow at high lung volumes. This is especially evident when the flows are expressed as a percentage of TLC or at absolute lung volumes. Although industrial bronchitis leads to a moderate increase in the RV, thereby suggesting that small airways are not entirely spared, it does not increase the TLC. The evidence, therefore, indicates that in a non-smoking subject with long-continued dust exposure who has cough and sputum plus a minor degree of large airways obstruction, both the symptoms and the pulmonary impairment can be assumed to be a consequence of occupational factors. In contrast, cigarette smoking, whether associated with bronchitis or not, leads to an increase in TLC (Figure 3). The inference to be drawn is that cigarette smoking leads to a loss of retractive forces in the lungs, and that this parenchymal change often occurs before the subject has symptoms. The presence of such structural changes in smokers does much to explain previously reported

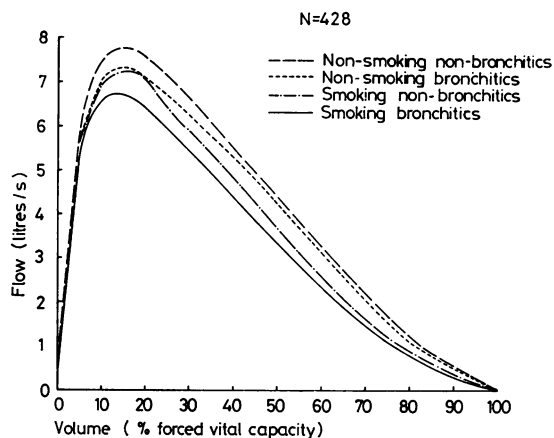


Fig. 2 Maximal expiratory flows of the four groups of coalminers expressed as a percentage of vital capacity. (Reproduced by permission of the Editor of the American Review of Respiratory Diseases.)

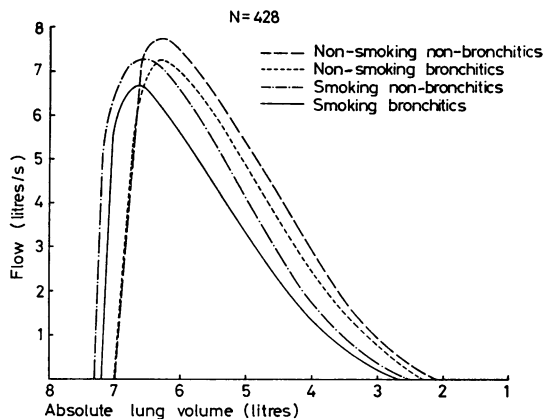


Fig. 3 Maximal expiratory flows of the four groups of miners expressed as absolute lung volumes. (Reproduced by permission of the Editor of the American Review of Respiratory Diseases.)

differences in morbidity and mortality in cigarette- and dust-induced bronchitis.

As a result of these studies, it is now possible to explain many of the anomalies previously noted and elaborated upon in the MRC statement. The 'very weak' relationship between bronchitis and coal workers' pneumoconiosis which has been discussed by Gilson (1970) can be explained by a difference in the size and site of deposition of inhaled particles (Muir, 1972). A similar lack of association between bronchitic symptoms and respirable dust has been observed not only in coal miners (Hyatt *et al.*, 1964; Enterline, 1967) but also in other dusty occupations

(McDonald *et al.*, 1972). Thus, those particles that are responsible for the development of coal workers' pneumoconiosis and silicosis, that is, the respirable fraction, are 0.5-5 μm in diameter. In contrast, provided that the subject is breathing through his nose, the majority of the particles deposited in the dead space range from 5 to 10 μm in diameter. Nevertheless, a small percentage of the respirable fraction will be deposited in the larger airways. If the subject is mouth-breathing, as so often happens for a limited time during and following exertion, many large particles between 10 and 20 μm in diameter will be deposited in the trachea and bronchi. The different sites of deposition are sufficient to explain the lack of association between coal workers' pneumoconiosis and bronchitis, and suggest that, in future when designing studies with the purpose of relating biological and environmental measurements, it would be wise to measure not only respirable but total dust. In addition, it is now evident that much of the reduction in ventilatory capacity that has been observed in dusty trades is a reflection of industrial bronchitis rather than of parenchymal involvement, namely pneumoconiosis. This would certainly seem to be true of coalminers and of the Vermont granite shed workers (Musk *et al.*, 1977). Recommendations to reduce the standards for respirable dust containing coal or silica are likely to be ineffective as far as the decrement attributable to industrial bronchitis is concerned. It would also seem that the original concern expressed by the Minister of Pensions, and which prompted the original MRC committee report, was justified, and that the decrement in FEV_{1.0} that is observed in men with category 0 or 1 simple pneumoconiosis of coal workers is indeed related to occupation if the man is a non-smoker. Those who maintain that, because the man has a category 2 or 3 pneumoconiosis, he therefore has been exposed to excessive dust, have also gone on to assume that the dust has caused functional impairment (Muir, 1977). In reality there is no evidence to suggest that this assumption is correct, because a similar decrement has also been observed in men with categories 0 and 1. Moreover, the dust that has caused the abnormal radiograph, that is the respirable fraction, is not the same dust that has caused the decrement in pulmonary function.

It now seems reasonable to accept the concept of industrial bronchitis as a cause of ventilatory impairment; however, its presence cannot be equated with disability. Any demonstrable abnormality of pulmonary function is an impairment but, plainly, most impairments do not affect a man's capacity to function normally or to carry out his job. Thus the demonstration of an abnormal closing volume or of frequency dependence of dynamic compliance in an

asymptomatic smoker is an indication that the man's pulmonary function is impaired, but one cannot regard such a person as being disabled. Moreover, when relating impairment to disability it is imperative to take into consideration the physical demands of the job; thus, the same impairment in different people does not necessarily lead to the same degree of disability.

One of the more important questions that remains unanswered is the reversibility of the reduction in ventilatory capacity induced by industrial bronchitis. While it is well recognised that the cough and sputum of cigarette smoker's bronchitis will clear up a few months after the subject stops smoking, this is not true of the physiological abnormalities associated with concomitant emphysema. An ideal opportunity to study the reversibility of industrial bronchitis exists in South Africa where the black gold miners, after a certain period of working in the mines, are given a prolonged spell off work before resuming their jobs in the mines.

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